complacent. As doctors or scientists we are asked to give opinions about a range of issues, not all of which lie within our area of expertise. We will probably also be asked about issues that are at the boundaries of medical and scientific knowledge and about which considerable uncertainty exists. It is incumbent on us to be aware of all the arguments and to be able to communicate the levels of uncertainty associated with them in an understandable fashion. We need to work with the media so that we remain in control of the debates, and, above all, we must preserve our professional credibility at a time when those around us are losing theirs.

JOHN HARRISON
Senior lecturer in occupational medicine

Department of Environmental and Occupational Medicine,
Medical School,
University of Newcastle,
Newcastle upon Tyne NE2 4HH

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... but members of the public make up their own minds about risks

EDITOR,—The government's recent statement on bovine spongiform encephalopathy has focused media interest on its potential impact on consumer behaviour.¹ The general public's response to media reports on "mad cow disease" and how this might have affected their behaviour already have not been well documented. In a survey among nurses 5% reported avoiding beef and a further 58% said that they would do so if cross species transmission was confirmed.²

Before the government's most recent statement we interviewed 155 members of the general public in three south London boroughs to assess their knowledge of bovine spongiform encephalopathy and how this related to changes in beef consumption. Twenty one general practitioners also completed the questionnaire.

Overall knowledge of bovine spongiform encephalopathy was good, with 147 of the members of the general public being aware of the disease. Their assessment of the risk of Creutzfeldt-Jakob disease was accurate, with 129 saying that it was either rare or extremely rare and 120 correctly estimating the number of deaths in Britain each year as 50 or fewer.³ Altogether 134 reported that the media were their source of information, with only five having consulted their general practitioner.

Eighty one (55%) members of the public thought that humans could get the disease by eating cow products, compared with 12 (57%) general practitioners. Sixty (41%) members of the public and 14 (67%) general practitioners had reduced their beef consumption. The public tended to avoid all beef products, whereas general practitioners were more likely to refrain selectively from eating offal products (39 of the public avoided all beef and 21 were selective, compared with four and 10 general practitioners respectively; $\chi^2=6.2$, $df=1$, $P=0.01$).

Generally the public were true to their beliefs and changed their behaviour accordingly. Forty (49%) of those who believed that transmission was possible had reduced their beef consumption, compared with 14 (29%) who were unsure about transmission and none who thought that transmission was impossible ($\chi^2=17.2$, $df=2$, $P=0.002$).

We also identified a subgroup of 59 people who had consistently changed their diet in response to previous media reports, such as the scares over salmonella and listeria in food. Of these, 34 (58%) had decreased their beef intake, compared with only 25 (28%) who had not previously changed their behaviour ($\chi^2=12.6$, $df=1$, $P=0.0004$).

Thus, before the recent statements, the general

public seemed to have picked up on the uncertainty about the scientific evidence portrayed in the media, made up their own minds, and acted accordingly. The public are more sophisticated than we give them credit for; public health messages should take this into account. If people are given absolute answers to questions for which the evidence is uncertain they seem not to believe the message.

K D GUNASEKERA, A I HAPGOOD, E L HARVEY,
T R M HAYFRON BENJAMIN, M S J JACHUCK,
A L N JACKSON, N P A KRONFELD, M I MANIKON,
C J MEARNES, N MUSHTAQ (final year medical students),
FIONA ADSHEAD (lecturer in public health)
Department of Public Health Sciences,
St George's Hospital Medical School,
London SW17 0RE

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Advisory committee's conclusion was based on "absence of any credible alternative"

EDITOR,—On 20 March the Spongiform Encephalopathy Advisory Committee in Britain issued a statement regarding 10 cases of Creutzfeldt-Jakob disease that showed a previously unrecognised and consistent pattern of disease.¹ The committee concluded: "in the absence of any credible alternative the most likely explanation at present is that these cases are linked to exposure to BSE [bovine spongiform encephalopathy] before the introduction of the SBO [specified bovine offal] ban in 1989." How can a committee of scientists come to a conclusion on the basis of no credible alternative? Why exposure to bovine spongiform encephalopathy and not to microwave ovens, high voltage power lines, or organophosphorus sheep dips?

These few words have caused an epidemic of hysteria across Europe and paralysed the British beef industry at home and abroad. Pharmaceutical companies are being besieged by patients concerned about the possible risks of medicines of bovine origin. For example, diabetic patients are asking whether they should continue to take beef insulin (in the case of the pharmaceutical company to which I am a medical adviser the insulins are made from pancreases from cattle outside the British Isles).

Another concern is that the suicide rate in British farmers—which is currently among the highest by occupation in Britain—will increase and that the increase will exceed the number of deaths from Creutzfeldt-Jakob disease over the coming months. All this when, in the committee's own words, "there is no direct evidence of a link."

ANNE WICKHAM
Consultant pharmaceutical physician

PO Box 246,
Canterbury,
Kent CT4 5RY

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Government policy has failed

EDITOR,—Several weeks ago two of us expressed concern about the credibility of government advice on public health.¹ The issue of bovine spongiform encephalopathy has confirmed the disbelief with which some government pronouncements on public health are regarded in Britain and worldwide. If this situation is not to be repeated lessons must be learnt from three failures of policy.

The deregulatory culture in Britain contributed to the failure to regulate recycling of sheep remains until 1989, although the Royal Commission on

Environmental Pollution had expressed concern about this in 1979.² Ministers have taken minimal action, which has consistently been based on the most optimistic scenarios.³ Deregulation is justified largely on financial grounds. Comparison of the total costs that will be incurred in Britain with those of more regulatory policies elsewhere shows that this is not tenable. Amazingly, the government confirmed its intention to relax further regulations on food hygiene immediately after a worldwide ban on the export of British beef was proposed.⁴ But food safety is not the only aspect of public health affected by deregulation: there are concerns about the effect of deregulation in, for example, the water, nuclear, and transport industries.

The second failure of policy was the way in which the Spongiform Encephalopathy Advisory Committee worked, and this was compounded by the absence from the committee, until recently, of specialist public health scientists. In the face of uncertainty, traditional methods of assessing evidence should be supplemented with techniques such as modelling of the consequences of best and worst case scenarios. Had the committee done this it might have placed less faith in the species barrier, given that many human infections originated in animals. It might also have questioned the official confidence in the implementation of precautions, given extensive evidence that they are not being taken. It also failed to appreciate how risk is perceived, which led to a patronising impression, characterised by the secretary of state's statement that "it is the public who are mad rather than the cows."

The third failure arises from the relation between the government and business in Britain. The Ministry of Agriculture, Fisheries and Food continues to resist proposals for an independent food standards agency,⁵ giving the impression that commercial interests come before those of the public. A national food policy council is also needed to coordinate the many interests involved. There are many other issues, such as the presence of pesticide residues in fruit and vegetables, on which consumer opinion will demand a source of authoritative, independent advice. The public is concerned that intensive farming has cut corners in cutting costs. On grounds of public health and confidence in governance this cannot continue.

MARTIN MCKEE
Reader in public health medicine
JENNIFER A ROBERTS
Senior lecturer in health economics

Health Services Research Unit,
London School of Hygiene and Tropical Medicine,
London WC1E 7HT

TIM LANG
Professor of food policy

Centre for Food Policy,
Wolfson School of Health Sciences,
Thames Valley University,
London W5 5RF

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Risk to human populations is remote

EDITOR,—Many commentaries on bovine spongiform encephalopathy have focused concern on recent unusual cases of Creutzfeldt-Jakob disease, an apparently important increase in the incidence of the disease, and statistical accretions in particular age and occupational groups. A link between the two diseases raises the possibility (even if not the probability) of risk to large numbers of people.¹ Indeed, the standardised mortality ratios for

deaths due to Creutzfeldt-Jakob disease reported to the Office of Population Censuses and Surveys during the 15 years 1979-93 show a significant excess in 1992 ($\chi^2=11.67$, $P<0.01$) and the interval 1987-93 ($\chi^2=4.29$, $P<0.05$). The age adjusted mortality rose by 30%, from 0.6 to 0.8 deaths per million.

Deaths from Creutzfeldt-Jakob disease in the two intervals 1979-85 and 1987-93 can also, however, be viewed as two samples taken from a common distribution, with the earlier sample being less complete than the later. The difference between the two distributions of cumulative mortality was minimal ($\chi^2=0.151$, $df=16$, $P>0.999$), and a two by two analysis of the proportion of deaths below age 45 during the two intervals (8.8% and 11% respectively) did not show a significant difference ($\chi^2=0.261$, $df=1$, $P=0.61$). The rise in overall mortality to date is therefore more likely to be attributable to increased awareness of Creutzfeldt-Jakob disease than to an increased risk of death.

Standard survival modelling techniques² indicate that there is a small subpopulation susceptible to death due to Creutzfeldt-Jakob disease, whose relatively high mean age at death could potentially be reduced by exposure to a triggering agent derived from cattle. In particular, the mortality is well fit by a multistage (Weibull) mortality distribution³ in which mortality rises as the seventh power of age (for all ages) and the risk of death is restricted to about 70 per million of the general population, or about 3500 people. Figure 1 compares the distributions of theoretical and observed mortality in 1979-85 ($\chi^2=1.42$, $df=19$, $P>0.999$) and 1987-93 ($\chi^2=0.329$, $df=19$, $P>0.999$) and also shows the projected effect of a 50% increase in exposure to risk. Declining mortality after age 67 can be explained by the reduction in the number of susceptible people.

Fig 1—Probability distribution of mortality from Creutzfeldt-Jakob disease in England and Wales during 1979-85 and 1987-93, best fitting Weibull distribution, and hypothetical curve based on 50% increase in risk

The earlier age at onset noted in cases with identified polymorphisms⁴ may merely reflect the difficulties of genetic analysis and detection of familial associations in late onset cases. While bovine spongiform encephalopathy may represent a risk to these select people, there is no evidence that it has done so.

STUART NEILSON
Director of medical information systems

Centre for the Study of Health, Sickness, and Disablement,
Brunel University,
Uxbridge,
Middlesex UB8 3PH

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Plasma lactate and blood cyanide in acute cyanide poisoning

Confounding factors should have been considered

EDITOR,—Frédéric J Baud and colleagues fail to consider the possibility of a confounding factor in their observed relation between plasma lactate and blood cyanide concentrations.¹ The patient with acute cyanide poisoning on whom they report had a cardiorespiratory arrest shortly after arriving in hospital, and resuscitation initially produced a very low arterial blood pressure. Further support was provided by a high dose adrenaline infusion. His condition improved with this treatment and infusion of hydroxycobalamin. Initial blood samples showed extremely high lactate concentrations, which were still raised at 10 hours.

The patient clearly had profound shock, including a period of circulatory standstill. In such circumstances, even in the absence of cyanide poisoning, blood lactate concentration will rise as a result of impaired tissue perfusion.² Inotropic support, while improving global haemodynamics (for example, normalising blood pressure), may not lead to resolution of lactic acidemia as there may be shunt across tissue beds, and oxygen use may not increase despite increases in oxygen delivery. In addition, if the liver is not perfused adequately lactate will not be cleared from the circulation. With improvement in global and regional perfusion, production of lactate will fall and clearance of lactate will increase, leading to a fall in plasma concentration. The data presented do not allow us to determine whether tissue perfusion was adequate during the first hours of treatment; the presence of high cyanide concentrations at tissue level produces a metabolic blockade, so that venous oxygen saturation is not a useful measure of global tissue perfusion.

These confounding factors blur the picture. While it is entirely reasonable to suspect a causal association between the time course of the lactic acidemia and that of the blood cyanide concentrations, the contribution to raised plasma lactate concentrations of cardiac arrest, shock, and subsequent inotropic support may be considerable. Similar observations in a patient who did not suffer cardiac arrest might be even more illuminating.

T M COOK
Locum senior registrar in anaesthesia

Southmead Hospital,
Bristol BS10 5NB

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Authors' reply

EDITOR,—We agree with T M Cook's analysis of the factors resulting in lactic acidosis in acute cyanide poisoning but not with the conclusion. We would like to focus our reply on the mechanisms of lactic acidosis induced by cyanide in mammals.

At a cellular level, short term exposure of cultured cells to cyanide rapidly induces a dramatic intracellular acidosis associated with increased lactate concentration. The blockade of cellular respiration through the inhibition of cytochrome aa3 by cyanide is considered to be the basic causative mechanism accounting for the increased production of lactate through the anaerobic pathway. Even in the case of intracellular acidosis it has now been shown that mechanisms other than increased production of lactate occur.¹

In mammals, however, numerous additional disturbances induced by cyanide eventually result

in the onset of severe lactic acidosis.^{2,3} We suggest that, as well as the blockade of cellular respiration, these disturbances should not be excluded, as proposed by Cook, but rather taken into account in any attempt to explain the basic mechanisms of lactic acidosis induced by cyanide in mammals. Acute cyanide poisoning induces sustained seizures, endogenous catecholamine release, increased blood glucose concentrations, and cardiovascular shock, which are all known to be associated with increased plasma lactate concentrations. As Cook points out, supportive treatment with an infusion of β mimetic agent is an additional cause of increased production of lactate. Furthermore, liver failure either through low cardiac output or through a direct effect of cyanide should result in a decrease in clearance of lactate from blood. These factors figure in previous reports of lactic acidosis in both experimental models and acute cyanide poisoning in humans. In rats the induction of lactic acidosis was recently shown to depend critically on the dose of cyanide.²

In conclusion, lactic acidosis induced by cyanide in mammals should be considered to be the result of numerous mechanisms at both cellular and organic levels whose respective contributions have not been assessed. Our case report suggests that, even in such a complex clinical setting, some relation seems to exist between plasma lactate and blood cyanide concentrations.

FRÉDÉRIC J BAUD
Professor of critical care medicine

ERIC BAVOUX
Attending physician in critical care medicine

STEPHEN W BORRON
Visiting researcher

JEROME HOFFMAN
Professor of emergency medicine, UCLA

Hôpital Fernand Widal,
75475 Paris Cedex 10,
France

ALAIN ASTIER
Chief of service

Laboratoire de Toxicologie,
Hôpital Henri Mondor,
Paris, France

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Vocational training schemes in south London have been improved

EDITOR,—Course organisers for vocational training schemes in general practice in south London have spent the past three years developing initiatives to deal with the problems described by Tess Harris and colleagues.¹ The four training schemes in south London (Guy's, King's, Lewisham, and St Thomas's) have cooperated to form the South London Organisation of Vocational Training Schemes; we have been supported by Lambeth, Southwark and Lewisham Health Commission and have obtained funding for educational initiatives.

The dearth of trainers, especially now that we are appointing more registrars in general practice, is more than compensated by the trainers' enthusiasm and skill and the quality of training that they offer to those who want to work in the inner city.

We have used funding provided for educational initiatives within the London Implementation Zone to purchase protected time for education. We have provided more variety, flexibility, and community focus for hospital based training in general practice. We have created innovative